

<b>L Numb r</b>	<b>Hits</b>	<b>Search Text</b>	<b>DB</b>	<b>Time stamp</b>
<b>1</b>	<b>0</b>	<b>"b n cell culture" sam "s rum fre "</b>	<b>USPAT; US-PGPUB; EP ; JPO; DERWENT</b>	<b>2003/04/01 13:50</b>
<b>2</b>	<b>11</b>	<b>"bone cell" same "serum free"</b>	<b>USPAT; US-PGPUB; EPO; JPO; DERWENT</b>	<b>2003/04/01 13:55</b>
<b>3</b>	<b>2</b>	<b>("5972703").PN.</b>	<b>USPAT; US-PGPUB; EPO; JPO; DERWENT</b>	<b>2003/04/01 13:55</b>
<b>4</b>	<b>2</b>	<b>("6152964").PN.</b>	<b>USPAT; US-PGPUB; EPO; JPO; DERWENT</b>	<b>2003/04/01 13:55</b>

130:63360

TI Serum-free cell growth medium for chondrocytes  
IN Luyten, Frank P.; Erlacher, Ludwig  
PA United States Dept. of Health and Human Services, USA  
SO PCT Int. Appl., 11 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9859035	A2	19981230	WO 1998-US12958	19980622
	WO 9859035	A3	19990318		
	W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
	RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG			
	AU 9879844	A1	19990104	AU 1998-79844	19980622
	US 2001039050	A1	20011108	US 2001-851921	20010509
PRAI	US 1997-50691P	P	19970625		
	WO 1998-US12958	W	19980622		
	US 1999-468562	B1	19991221		

AB A chem. defined-serum free growth medium for the (in vitro) and ex vivo of cells and cell lines. The medium consists of about a one to one ratio (vol./vol.) of two basal growth media contg. .alpha.-ketoglutarate, insulin, transferrin, selenium, bovine serum albumin, linoleic acid, ceruloplasmin, cholesterol, phosphatidylethanolamine, .alpha.-tocopherol acid succinate, reduced glutathione, taurine, triiodothyronine, hydrocortisone, parathyroid hormone, L-ascorbic acid 2-sulfate, .beta.-glycerophosphate, PDGF, EGF and FGF. Chondrocytes, when cultured in this medium in the presence of a cartilage derived morphogenetic protein or bone morphogenetic protein, retain their cartilaginous phenotype. This invention also provides a method of repairing a joint surface defect.

DUPLICATE 37

AN 1996:376354 BIOSIS

DN PREV199699098710

TI Ontogenesis of IGF regulation of longitudinal bone growth in rat metatarsal rudiments cultured in serum-free medium.

AU Coxam, V. (1); Miller, M. A.; Bowman, M. B.; Miller, S. C.

CS (1) Metabolisme Mineral Osteogenese Croissance Metabolismes Herbivores, INRA Theix, 63122 Saint Genes Champanelle France

SO Archives of Physiology and Biochemistry, (1996) Vol. 104, No. 2, pp. 173-179.

ISSN: 1381-3455.

DT Article

LA English

AB The aim of this study was to compare the effect of two cytokines, IGF-I and IGF-II on skeletal development in the rat. The three medial metatarsal rudiments were dissected out from fetuses at days 19, 20 or 21 of gestation and from newborns at days 1, 3, 6 and 9 after birth, then grown in serum-free MEM medium at 37 degree C and 5% CO-2 in air. From day 19 of gestation to the end of experiment, longitudinal bone growth (mm) was significantly increased by IGF-I ( $2.975 \pm 0.050$ ) and IGF-II ( $2.530 \pm 0.062$ ), compared to controls ( $2.188 \pm 0.060$ ). In the same way, the width (mm) at the last experimental day was  $0.360 \pm 0.010$  in IGF-I- and  $0.327 \pm 0.008$  in IGF-II-treated bones, respectively (vs  $0.313 \pm 0.012$  in controls). Mineralization was also stimulated under both growth factors (length of the calcified diaphysis (mm) :  $0.691 \pm 0.019$  in IGF-I - and  $0.446 \pm 0.017$  in IGF-II-treated bones; vs  $0.383 \pm 0.024$  in controls). IGF-I and IGF-II (but to a lesser extent) stimulation was due to an increased DNA synthesis (3H-thymidine uptake) as well as protein anabolism (incorporated proline). In addition, cartilage activity (35S captation) and mineralization (45Ca fixed) were involved in the action of these cytokines. An age dependency of bone response to IGFs was pointed out, the effect being higher during the fetal period than after birth. In conclusion, our results raise the possibility that IGF-II, as well as IGF-I, is involved in the control of osteogenesis.

93250527 PubMed ID: 7683536

TI Insulin-like growth factor binding proteins in bone cell regulation.  
AU Mohan S  
CS Department of Medicine, Loma Linda University, CA.  
NC AR 31062 (NIAMS)  
SO GROWTH REGULATION, (1993 Mar) 3 (1) 67-70. Ref: 27  
Journal code: 9106990. ISSN: 0956-523X.  
CY SCOTLAND: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
General Review; (REVIEW)  
(REVIEW, TUTORIAL)  
LA English  
FS Priority Journals  
EM 199306  
ED Entered STN: 19930618  
Last Updated on STN: 19960129  
Entered Medline: 19930609  
AB Recent studies emphasize that 1) IGFs are important local regulators of bone formation and 2) IGFBPs are important regulators of the biological actions of IGFs in bone. The importance of IGFs is shown by the finding that 40-50% of basal bone cell proliferation could be blocked by inhibiting the actions of IGFs produced endogenously by bone cells in **serum free culture**. In addition, IGFs are the most abundant growth factors stored in bone and are produced by bone cells. ~~Recent studies suggest that IGFs are fixed in bone by means of IGFBP-5 which binds with high affinity to both hydroxyapatite and IGFs.~~ Upon release from this storage depot, IGFs and IGFBP-5 are thought to act in the coupling of bone formation to bone resorption in a delayed paracrine manner (i.e. previously fixed IGF+IGFBP-5 complex is released in a bioactive form during bone resorption to stimulate new bone formation). In addition to IGFBP-5, human bone cells in culture have also been shown to produce other IGFBPs, some of which modulate IGF actions in either positive or negative manner. In addition, recent studies also demonstrate that local and systemic effectors of bone formation may regulate the actions of acutely synthesized IGFs (autocrine/paracrine actions) in bone cell microenvironment in a tissue specific manner by modulating the type and amount of IGFBPs produced by bone cells at a local site of bone.

L3 ANSWER 92 OF 181 MEDLINE DUPLICATE 54  
 AN 93245078 MEDLINE  
 DN 93245078 PubMed ID: 1338704  
 TI Effects of prostaglandin E2 on growth and function of osteoblasts in human cell culture in vitro.  
 AU Peng Q  
 CS Tianjin of Endocrinology Institute.  
 SO CHUNG-HUA I HSUEH TSA CHIH [CHINESE MEDICAL JOURNAL], (1992 Nov) 72 (11) 667-9, 702.  
 Journal code: 7511141. ISSN: 0376-2491.  
 CY China  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA Chinese  
 FS Priority Journals  
 EM 199305  
 ED Entered STN: 19930618  
 Last Updated on STN: 19930618  
 Entered Medline: 19930528  
 AB The effects of prostaglandin E 2 (PGE2) on the differentiation and proliferation of osteoblasts (human fetal **bone**-cells) **cultured in serum-free** medium were investigated by assays of alkaline phosphatase (ALP) activity, intracellular cyclic AMP level and collagen synthesis in the cells. The results suggested that PGE2 in physiologic concentration stimulated the differentiation of osteoblasts in vitro, and might be involved in bone formation in vivo.

L3 ANSWER 97 OF 181 MEDLINE  
AN 93239960 MEDLINE  
DN 93239960 PubMed ID: 1300341  
TI Osteoblastic control of osteoclast **bone** resorption in a  
**serum-free** co-culture system. Lack of effect  
of parathyroid hormone.  
AU Teti A; Grano M; Colucci S; Zambonin Zallone A  
CS Institute of Human Anatomy, School of Pharmacy, University of Bari, Italy.  
SO JOURNAL OF ENDOCRINOLOGICAL INVESTIGATION, (1992) 15 (9 Suppl 6) 63-8.  
Journal code: 7806594. ISSN: 0391-4097.  
CY Italy  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 199305  
ED Entered STN: 19930611  
Last Updated on STN: 19930611  
Entered Medline: 19930527

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ANSWER 109 OF 181 MEDLINE

AN 93082282 MEDLINE

DN 93082282 PubMed ID: 1842348

TI Effects of recombinant human insulin-like growth factor I and II (IGF-I/-II) and growth hormone (GH) on the growth of normal adult human osteoblast-like cells and human osteogenic sarcoma cells.

AU Scheven B A; Hamilton N J; Fakkeldij T M; Duursma S A

CS University Hospital Utrecht, Research Group for Bone Metabolism, The Netherlands.

SO GROWTH REGULATION, (1991 Dec) 1 (4) 160-7.

Journal code: 9106990. ISSN: 0956-523X.

CY SCOTLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199301

ED Entered STN: 19930129

Last Updated on STN: 19970203

Entered Medline: 19930107

AB Recombinant GH and IGF-I/-II were studied for their capacity to directly influence the growth of human **bone** cells maintained under defined **serum-free** conditions. Normal human osteoblast-like cell (HOB) **cultures** were established from trabecular bone explants obtained from adult human femoral head samples. IGF-I and IGF-II as well as GH stimulated the growth of the HOB cultures in a dose-dependent manner. Growth stimulatory effects were also found using the human osteogenic sarcoma cell line, SaOS-2. IGF-I and -II were powerful enhancers of the SaOS-2 cell growth and their effects greatly exceeded GH effects on these cells. The role of endogenously produced IGFs was studied using a specific monoclonal antibody to IGF-I having a partial cross-reactivity with IGF-II (sm1.2B). The IGF-I stimulated HOB growth was completely neutralised by sm1.2B to the level of control+antibody which in general showed a slight stimulation compared to controls without the antibody. Interestingly, sm1.2B was not able to interfere with the action of GH on the HOB suggesting that GH effects may be attributed to an action independent of endogenous IGF-I/-II. Unlike the HOB, SaOS-2 cells were strongly inhibited by sm1.2B in control medium indicating an autocrine role of IGF-I/-II in osteosarcoma cell growth. Sm1.2B completely neutralised the stimulatory effects of IGF-I and IGF-II on the SaOS-2 cells. Moreover, GH effects on the osteogenic sarcoma cells were abolished by the anti-IGF antibody showing that GH was acting via endogenously produced IGFs. (ABSTRACT TRUNCATED AT 250 WORDS)

L3 ANSWER 110 OF 181 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

L3 ANSWER 115 OF 181 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.  
AN 1992:520217 BIOSIS  
DN BA94:128292  
TI EFFECT OF 3 5 3 TRIIODOTHYRONINE ON GROWTH AND FUNCTION OF OSTEOLASTS IN  
HUMAN CELL CULTURE IN-VITRO.  
AU PENG Q; ET AL  
CS TIANJIN ENDOCRINOL. INST.  
SO TIANJIN MED J, (1990) 18 (10), 589-591.  
CODEN: TIYADG. ISSN: 0253-9896.  
FS BA; OLD  
LA Chinese  
AB The effect of 3,5,3'-triiodothyronine (T3) on the differentiation and  
proliferation of osteoblasts (human fetal **bone**-cells)  
**cultured** in **serum-free** medium was investigated  
by assays of alkaline phosphatase (ALP) activity, intracellular cyclic AMP  
level and collagen synthesis in the cell. The result suggested that T3 in  
physiologic concentration stimulated the differentiation of osteoblasts in  
vitro, and might be involved in bone formation in vivo.

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L3 ANSWER 125 OF 181 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.  
AN 1990:67899 BIOSIS  
DN BA89:35725  
TI EFFECT OF PROSTAGLANDIN E-1 AND PROSTAGLANDIN F-2-ALPHA ON BONE FORMATION  
OF CULTURED CHICK EMBRYONIC BONE WITH SPECIAL REFERENCE TO THE ALKALINE  
PHOSPHATASE ACTIVITY.  
AU UEDA S  
CS FIRST DEP. ORAL AND MAXILLOFACIAL SURGERY, OKAYAMA UNIV. DENT. SCH.,  
OKAYAMA 700, JPN.  
SO OKAYAMA IGAKKAI ZASSHI, (1989) 101 (7-8), 763-770.  
CODEN: OIZAAB. ISSN: 0030-1558.  
FS BA; OLD  
LA Japanese  
AB The effects of prostaglandin E1 (PGE1) and prostaglandin F2.alpha.  
(PGF2.alpha.1) on bone formation were investigated with femur and tibia  
from 10-day chick embryo. When the bones were **cultured** in  
**serum free** medium, PGE1 (10nM) caused a significant  
increase of **bone** alkaline phosphatase activity and a slight  
increase in bone protein content, whereas PGF2.alpha. (100nM)  
significantly decreased the enzyme activity. On the other hand, in a  
medium containing 20% serum, PGE1 did not affect the enzyme activity at  
10nM, but slightly increased the activity at 1nM. No effect of PGE1 on bone  
calcium content was observed in a concentration ranging from 0.1 nM to 100  
nM. Thus, ~~PGE1 was likely to stimulate bone formation at 10nM; whereas~~  
PGF2.alpha.2 suppressed formation at 100nM.

L3 ANSWER 126 OF 181 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.  
 DUPLICATE 78  
 AN 1990:44744 BIOSIS  
 DN BA89:22108  
 TI MECHANICAL STIMULATION BY INTERMITTENT COMPRESSION STIMULATES SULFATE IN  
 CORPORATION AND MATRIX MINERALIZATION IN FETAL MOUSE LONG-BONE RUDIMENTS  
 UNDER SERUM-FREE CONDITIONS.  
 AU BAGI C; BURGER E H  
 CS UNIV. UTAH, RADIOBIOL. DIV., BLD. 586, SALT LAKE CITY, UTAH 84112.  
 SO CALCIF TISSUE INT, (1989) 45 (6), 342-347.  
 CODEN: CTINDZ. ISSN: 0171-967X.  
 FS BA; OLD  
 LA English  
 AB Mechanical stimulation evoked by intermittent hydrostatic compression (IC)  
 in a closed culture system has been shown to stimulate calcification of  
 fetal long-bone rudiments in the presence of serum [6]. We have studied  
 effects of IC on sulfate metabolism and matrix mineralization under  
**serum-free** conditions, in short-term (24 hours)  
**cultures** of mineralizing long-bone rudiments in alpha  
 minimum essential medium (MEM) + 0.2% bovine serum albumen (BSA). Exposure  
 to IC for 24 hours stimulated radiosulfate incorporation into the  
 papain-digestible pool in the noncalcifying epiphyses and, to a larger  
 extent, in the calcifying diaphysis. The percentage release of 35S from  
 prelabeled rudiments was stimulated in the epiphyses, but inhibited in the  
 diaphyses. The changes in sulfate metabolism of matrix mineralization, in  
 hypertrophic cartilage, and the diaphyseal bone collar were judged from  
 the increase in length of the diaphysis. This study shows that under  
 serum-free conditions, mechanical stimulation by IC increases sulfate  
 content while stimulating mineralization in calcifying cartilage of fetal  
 long-bone rudiments. Mechanical stimulation seems to be an important  
 regulator of cartilage calcification.

L3 ANSWER 129 OF 181 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.  
 DUPLICATE 81  
 AN 1989:132791 BIOSIS  
 DN BA87:67444  
 TI MURINE OSTEOBLASTS RELEASE BONE-RESORBING FACTORS OF HIGH AND LOW  
 MOLECULAR WEIGHTS STIMULATION BY MECHANICAL DEFORMATION.  
 AU SANDY J R; MEGHJI S; SCUTT A M; HARVEY W; HARRIS M; MEIKLE M C  
 CS ORAL SURGERY RES. LAB., EASTMAN DENT. HOSP., 256 GRAY'S INN ROAD, LONDON  
 WC1X 8LD, ENGLAND.  
 SO BONE MINER, (1989) 5 (2), 155-168.  
 CODEN: BOMIET. ISSN: 0169-6009.  
 FS BA; OLD  
 LA English  
 AB Murine calvarial osteoblasts in monolayer culture were found to  
 constitutively produce bone-resorbing factors; mechanical deformation  
 significantly increased the synthesis and/or release of these factors. In  
 short-term cultures (2 h) the resorptive activity was largely dialysable,  
 indicating a relative molecular mass (Mr) less than 2000. Intermittent  
 mechanical deformation stimulated the synthesis of these low Mr factors  
 irrespective of serum conditions. Continuous deformation, however, was  
 without effect. When the culture period was extended to 24 h, bone  
 resorptive activity was stimulated by both intermittent and continuous  
 deformation in the presence of 10% serum. This activity was dialysable.  
~~Over this same period in cultures with 2% serum, intermittent~~  
 deformation also produced a non-dialysable bone-resorbing  
 factor. We also cultured osteoblasts for 72 h in serum  
 -free conditions and deformed the cells intermittently.  
 Fractionation of the medium by high pressure liquid chromatography (HPLC)  
 resolved three peaks of bone resorptive activity: peak I (Mr 50-60,000);  
 peak II (Mr 5-20,000); and peak III (Mr < 1000). Only peaks II and III  
 were stimulated by mechanical deformation. These bone-resorbing factors  
 remain as yet poorly characterized, but none of the activity in the HPLC  
 fractions was attributable to interleukin-I or prostaglandin E2.

84258682 MEDLINE

DN 84258682 PubMed ID: 6430514  
TI Bone-derived factors active on bone cells.  
AU Mohan S; Linkhart T; Farley J; Baylink D  
NC AM 31061 (NIADDK)  
AM 31062 (NIADDK)  
SO CALCIFIED TISSUE INTERNATIONAL, (1984) 36 Suppl 1 S139-45.  
Journal code: 7905481. ISSN: 0171-967X.  
CY GERMANY, WEST: Germany, Federal Republic of  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals; Space Life Sciences  
EM 198409  
ED Entered STN: 19900320  
Last Updated on STN: 19970203  
Entered Medline: 19840918  
AB Effects of systemic calcium regulating hormones have been studied extensively, yet mechanisms of bone volume regulation at the local level are poorly understood. Our laboratory has reported evidence for two locally mediated processes of bone volume regulation which function independently of systemic control: (1) coupling of bone formation and resorption and (2) repletion of resorbed bone. These local regulatory mechanisms have been shown to occur in vivo and in vitro. We have reported that embryonic chick tibiae in **culture**, stimulated to resorb, release a factor in the ~~serum-free culture~~ medium that stimulates **bone** cell proliferation and **bone** matrix formation in vitro. We have postulated that this factor could be involved in the coupling mechanism. Subsequently, a similar factor which stimulates bone cell proliferation, collagen synthesis and bone formation in vitro was extracted from embryonic and adult bones. The factor partially purified from human bone, designated as human skeletal growth factor, has molecular weight, heat sensitivity and biological activity similar to the factor found in bone conditioned medium. Many other biologically active factors have also been extracted from bone cells or demineralized bone by different laboratories. Their actions on bone cells range from chemotactic to mitogenic. These recently discovered bone factors emphasize that there is important regulation of bone metabolism at the local level.

L3 ANSWER 173 OF 181 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.  
DUPLICATE 113  
AN 1983:190859 BIOSIS  
DN BA75:40859  
TI PURIFICATION OF A SKELETAL GROWTH FACTOR FROM HUMAN BONE.  
AU FARLEY J R; BAYLINK D J  
CS RES. SERVICE, JERRY L. PETTIS MEMORIAL VETERANS' HOSPITAL, LOMA LINDA, CA  
92357.  
SO BIOCHEMISTRY, (1982) 21 (14), 3502-3507.  
CODEN: BICHAW. ISSN: 0006-2960.  
FS BA; OLD  
LA English  
AB A skeletal growth factor was isolated and purified from demineralized  
human bone matrix. A dose of 6 .mu.g/ml of the purified factor  
significantly increased the proliferation rate of embryonic chick  
**bone cells in serum-free culture**  
(292% of controls, P < 0.0001) but had no effect on embryonic chick skin  
cells plated at the same initial density. The factor is sensitive to  
inactivation by trypsin and urea, but not by collagenase, 20% butanol, or  
1% mercaptoethanol. It is also resistant to inactivation by heat (stable  
for 15 min at 75.degree. C) and extremes of pH (stable for 30 min at  
4.degree. C from pH 2.5 to 10.0). Purification of the active factor by  
selective heat and acid precipitations, molecular sieve column  
chromatography, and preparative-polyacrylamide-gel electrophoresis  
provided a material that was homogeneous by the criteria of high pressure  
liquid chromatography, polyacrylamide gel electrophoresis, and isoelectric  
focusing. The apparent MW is 83,000. The purified factor increases bone  
cell proliferation at doses comparable to other mitogens: 0.3 .mu.g/ml  
(3.6 nM) significantly increases DNA synthesis to 231% of controls (P <  
0.001). The purified factor was also active on cultured embryonic chick  
bones, enhancing the growth rate of tibiae and femurs, as measured by  
increased dry weight (185% of controls, P < 0.025) and [3H]proline  
incorporation (164% of control, P < 0.001), respectively.

84258682 PubMed ID: 6430514

TI Bone-derived factors active on bone cells.

AU Mohan S; Linkhart T; Farley J; Baylink D

NC AM 31061 (NIADDK)

AM 31062 (NIADDK)

SO CALCIFIED TISSUE INTERNATIONAL, (1984) 36 Suppl 1 S139-45.

Journal code: 7905481. ISSN: 0171-967X.

CY GERMANY, WEST: Germany, Federal Republic of

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals; Space Life Sciences

EM 198409

ED Entered STN: 19900320

Last Updated on STN: 19970203

Entered Medline: 19840918

AB Effects of systemic calcium regulating hormones have been studied extensively, yet mechanisms of bone volume regulation at the local level are poorly understood. Our laboratory has reported evidence for two locally mediated processes of bone volume regulation which function independently of systemic control: (1) coupling of bone formation and resorption and (2) repletion of resorbed bone. These local regulatory mechanisms have been shown to occur in vivo and in vitro. We have reported that embryonic chick tibiae in culture, stimulated to resorb, release a factor in the serum-free culture medium that stimulates bone cell proliferation and bone matrix formation in vitro. We have postulated that this factor could be involved in the coupling mechanism. Subsequently, a similar factor which stimulates bone cell proliferation, collagen synthesis and bone formation in vitro was extracted from embryonic and adult bones. The factor partially purified from human bone, designated as human skeletal growth factor, has molecular weight, heat sensitivity and biological activity similar to the factor found in bone conditioned medium. Many other biologically active factors have also been extracted from bone cells or demineralized bone by different laboratories. Their actions on bone cells range from chemotactic to mitogenic. These recently discovered bone factors emphasize that there is important regulation of bone metabolism at the local level.

L3 ANSWER 42 OF 52 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
30

AN 1989:132791 BIOSIS

DN BA87:67444

TI MURINE OSTEOBLASTS RELEASE BONE-RESORBING FACTORS OF HIGH AND LOW  
MOLECULAR WEIGHTS STIMULATION BY MECHANICAL DEFORMATION.

AU SANDY J R; MEGHJI S; SCUTT A M; HARVEY W; HARRIS M; MEIKLE M C

CS ORAL SURGERY RES. LAB., EASTMAN DENT. HOSP., 256 GRAY'S INN ROAD, LONDON  
WC1X 8LD, ENGLAND.

SO BONE MINER, (1989) 5 (2), 155-168.

CODEN: BOMIET. ISSN: 0169-6009.

FS BA; OLD

LA English

AB Murine calvarial osteoblasts in monolayer culture were found to  
constitutively produce bone-resorbing factors; mechanical deformation  
significantly increased the synthesis and/or release of these factors. In  
short-term cultures (2 h) the resorptive activity was largely dialysable,  
indicating a relative molecular mass (Mr) less than 2000. Intermittent  
mechanical deformation stimulated the synthesis of these low Mr factors  
irrespective of serum conditions. Continuous deformation, however, was  
without effect. When the culture period was extended to 24 h, bone  
resorptive activity was stimulated by both intermittent and continuous  
deformation in the presence of 10% serum. This activity was dialysable.  
~~Over this same period in cultures with 2% serum, intermittent deformation~~  
also produced a non-dialysable **bone-resorbing** factor. We also  
**cultured** osteoblasts for 72 h in **serum-free**  
conditions and deformed the cells intermittently. Fractionation of the  
medium by high pressure liquid chromatography (HPLC) resolved three peaks  
of bone resorptive activity: peak I (Mr 50-60,000); peak II (Mr 5-20,000);  
and peak III (Mr < 1000). Only peaks II and III were stimulated by  
mechanical deformation. These bone-resorbing factors remain as yet poorly  
characterized, but none of the activity in the HPLC fractions was  
attributable to interleukin-I or prostaglandin E2.